Journal Announcement: USGRDR6914
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Display 4/3/21 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

12517406 21341116 PMID: 11447764
Immunity to oncogenic **human papillomaviruses.**
Konya J; Dillner J
Laboratory of Tumor Virus Epidemiology, Microbiology and Tumor Biology Center, Karolinska Institute, S-17177 Stockholm, Sweden.
Advances in cancer research (United States) 2001, 82 p205-38, ISSN 0065-230X Journal Code: 0370416
Languages: ENGLISH
Document type: Journal Article; Review; Review, Academic
Record type: Completed

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DIALOG(R)File 154:MEDLINE(R)

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Languages: ENGLISH
Document type: Journal Article; Review; Review, Academic
Record type: Completed
Subfile: INDEX MEDICUS
The establishment of **human papillomavirus** (**HPV**) infection as a major cause of several human cancer forms, notably cervical cancer, has spurred development of prophylactic and/or therapeutic **HPV vaccines** for prevention of cervical neoplasia. Knowledge of the immunity to **HPV** forms the basis for such endeavors. METHOD: A literature review of humoral and cellular immunity to **HPV**. The

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overview on human leukocyte antigen (HLA) and cervical cancer was expanded to a formal metaanalysis, where relevant articles were located by Medline search and citation analysis and graded by preassigned quality criteria on study design. RESULTS: The antibody response to the **HPV** particle is dominated by a neutralizing antibody response to a typespecific, conformationally dependent immunodominant epitope. **Vaccines** based on viral particles lacking the viral genome (virus-like particles, VLPs) have been highly successful in preventing and treating **HPV** infection in several animal model systems. In humans, the serum antibody response to

VLPs is stable over time, also after the **HPV** infection has been cleared, resulting in **HPV** serology being used as a marker of cumulative **HPV** exposure in spite of the fact that a significant proportion of **HPV**-exposed subjects **fail** to seroconvert. More than 90% of **HPV** infections will clear spontaneously. The factors that determine whether an **HPV** infection is cleared or persists and increases the risk for cancer are not known, but cellular immunity is implicated. Several HLA class II haplotypes are associated with cervical cancer: DQw3 increases and DR13 decreases the risk for cervical cancer in

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general (odds ratios (OR) and 95% confidence intervals (CI): 1.25(1.15-1.37) and 0.69 (0.56-0.85), respectively); DR15 increases the risk for **HPV16**-carrying cancer (OR: 1.47; CI: 1.20-1.81); and DR7 may be either protective or increase the risk. Most cervical cancers have downregulated the expression of at least one HLA class I antigen, whereas class II expression is increased in infected epithelium. A Th2 cytokine profile is associated with progression to cervical cancer. **HPV** -antigen-specific proliferative responses have been detected in many studies, although it is not entirely clear whether these responses are **HPV** type specific or may be cross-reactive between **HPV** types. Specific cytotoxic T lymphocyte (CTL) responses were originally reported in only a minority of infected subjects, typically cancer patients, but with advancing technology, specific CTLs can be stimulated from about half of the women with **HPV** -carrying disease. In animal model systems, CTL responses can mediate clearance. CONCLUSION: The antibody response to **HPV** is a mediator of type-specific protective immunity, which forms the basis for prophylactic **vaccine** candidates. The cellular immunity to **HPV** is implicated as an important factor in cervical

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DIALOG(R)File 154:MEDLINE(R)
carcinogenesis, but the main targets and types of responses that mediate **HPV** clearance are not established. (175 Refs.)
Tags: Female; Human; Support, Non-U.S. Gov't
Descriptors: Cervix Neoplasms--virology--VI; ***Papillomavirus, Human**--immunology--IM; *Papovaviridae Infections--immunology--IM; *Tumor Virus Infections--immunology--IM; Antibodies, Viral--immunology--IM; Antibody Formation; Cervix Neoplasms--immunology--IM; HLA Antigens --analysis--AN; Immunity, Cellular; Papovaviridae Infections--virology--VI; Tumor Virus Infections--virology--VI
CAS Registry No.: 0 (Antibodies, Viral); 0 (HLA Antigens)
Record Date Created: 20010712

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Display 4/3/22 (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

11463598 21214158 PMID: 11313416
Improving **vaccine** potency through intercellular spreading and enhanced MHC class I presentation of antigen.
Hung CF; Cheng WF; Chai CY; Hsu KF; He L; Ling M; Wu TC
Department of Pathology, Johns Hopkins Medical Institutions, Baltimore, MD 21205, USA.